I CLAIM:

1. A method for simultaneously screening a plurality of candidate compounds for their ability to inhibit formation of a crystalline structure of a selected biomolecule that is endogenous to the human body, wherein *in vivo* formation of the crystalline structure is an adverse event resulting in at least one medical pathology selected from diseases, disorders, and other undesirable physiological conditions, the method comprising:

(a) providing a combinatorial library of a plurality of different candidate compounds each attached to a different site on a substrate;

(b) contacting the library of candidate compounds with the selected biomolecule under conditions effective to facilitate formation of said crystalline structure in the absence of any inhibitors;

(c) identifying candidate compounds for which the biomolecule has affinity by determining which candidate compounds have become physically associated with the biomolecule during step (b); and

(d) selecting the candidate compounds identified in step (c) as potential inhibitors of *in vivo* formation of the crystalline structure of the biomolecule.

2. The method of claim 1, wherein the different candidate compounds are selected so that they are structurally similar but nonidentical to the biomolecule.

3. The method of claim 2, wherein formation of the crystalline structure comprises formation of fibrils.

4. The method of claim 2, wherein formation of the crystalline structure comprises formation of ocular cataracts.

5. The method of claim 1, wherein the biomolecule is selected from the group consisting of peptidic molecules, sterols, uric acid, uric acid salts, and calcium salts.

6. The method of claim 5, wherein the biomolecule is a peptidic molecule.

7. The method of claim 6, wherein the candidate compounds are independently selected from the group consisting of:

(a) oligopeptide fragments contained within the peptidic molecule; and

(b) analogs of the peptidic molecule wherein the peptidic molecule is modified by (i) substitution of one or more amino acids, (ii) deletion of one or more amino acids, (iii) insertion of one or more amino acids, (iv) an N-terminal modification, (v) a C-terminal modification, or combinations thereof.

8. The method of claim 3, wherein the biomolecule is a peptidic molecule.

9. The method of claim 8, wherein the candidate compounds are independently selected from the group consisting of:

(a) oligopeptide fragments contained within the peptidic molecule; and

(b) analogs of the peptidic molecule wherein the peptidic molecule is modified by (i) substitution of one or more amino acids, (ii) deletion of one or more amino acids, (iii) insertion of one or more amino acids, (iv) an N-terminal modification, (v) a C-terminal modification, or combinations thereof.

10. The method of claim 4, wherein the biomolecule is selected from the group consisting of peptidic molecules, sterols, uric acid, uric acid salts, and calcium salts.

11. The method of claim 10, wherein the biomolecule is a peptidic molecule.

12. The method of claim 11, wherein the candidate compounds are independently selected from the group consisting of:

(a) oligopeptide fragments contained within the peptidic molecule; and

(b) analogs of the peptidic molecule wherein the peptidic molecule or oligopeptide fragments thereof are modified by (i) substitution of one or more amino acids, (ii) deletion of one or more amino acids, (iii) insertion of one or more amino acids, (iv) an N-terminal modification, (v) a C-terminal modification, or combinations thereof.

13. The method of claim 8, wherein the biomolecule is selected from the group consisting of amyloid and amyloid-β.

14. The method of claim 13, wherein the biomolecule is amyloid-β.

15. The method of claim 14, wherein the amyloid-β is selected from the group consisting of amyloid-β (1-40), amyloid-β (1-42), and amyloid-β (1-43).

16. The method of claim 14, wherein the candidate compounds are independently selected from the group consisting of:

(a) oligopeptide fragments contained within amyloid-β; and

(b) amyloid-β analogs wherein amyloid-β or oligopeptide fragments thereof are modified by (i) substitution of one or more amino acids, (ii) deletion of one or more amino acids, (iii) insertion of one or more amino acids, (iv) an N-terminal modification, (v) a C-terminal modification, or combinations thereof.

17. The method of claim 16, wherein the candidate compounds are oligopeptide fragments contained within amyloid-β.

18. The method of claim 8, wherein the biomolecule is prion protein (PrP) or an oligopeptide fragment thereof.

19. The method of claim 18, wherein candidate compounds are independently selected from the group consisting of PrP analogs wherein PrP or oligopeptide fragments thereof are modified by (i) substitution of one or more amino acids, (ii) deletion of one or

more amino acids, (iii) insertion of one or more amino acids, (iv) an N-terminal modification, (v) a C-terminal modification, or combinations thereof.

20. The method of claim 18, wherein the biomolecule is PrP.

21. The method of claim 18, wherein the biomolecule is a PrP fragment.

22. The method of claim 21, wherein the PrP fragment is PrP96-111.

23. The method of claim 8, wherein the biomolecule is fibrillar collagen.

24. The method of claim 8, wherein the biomolecule is fibrillin.

25. The method of claim 6, wherein the biomolecule is a lenticular protein.

26. The method of claim 25, wherein the biomolecule is selected from beta, gamma, alpha crystallins, and combinations thereof.

27. The method of claim 26, wherein the biomolecule is selected from beta and gamma crystallins.

28. The method of claim 26, wherein the biomolecule is comprised of a mixture of beta and gamma crystallins.

29. The method of claim 27, wherein the biomolecule is selected from the group consisting of β_H-crystallins, γS-crystallins, γC-crystallins, γD-crystallins, and combinations thereof.

30. The method of claim 6, wherein the biomolecule is cystic fibrosis transmembrane conductance regulator ("CFTR") protein.

31. The method of claim 6, wherein the biomolecule is a Charcot-Leyden protein.

32. The method of claim 6, wherein the biomolecule is selected from the group consisting of cystine, hemoglobin, hematoidin, cryoglobulins, and immunoglobulins.

33. The method of claim 5, wherein the biomolecule is a sterol.

34. The method of claim 33, wherein the biomolecule is cholesterol, cholesterol monohydrate, or a cholesteryl ester.

35. The method of claim 5, wherein the biomolecule is a calcium salt.

36. The method of claim 35, wherein the calcium salt is calcium oxalate, calcium oxalate monohydrate, hydroxyapatite, octacalcium phosphate, tricalcium phosphate, calcium hydrogen phosphate dihydrate, calcium pyrophosphate, or calcium pyrophosphate dihydrate.

37. The method of claim 10, wherein the biomolecule is uric acid or a salt thereof.

38. The method of claim 37, wherein the biomolecule is monosodium urate or monosodium urate monohydrate.

39. The method of claim 1, wherein the biomolecule is labeled with a detectable label, and step (c) is carried out by detecting the presence of the label at any of the different sites on the substrate.

40. The method of claim 39, wherein the label is a fluorescent label, and is detected using fluorescence spectroscopy.

41. The method of claim 39, wherein the label is detectable using ultraviolet spectroscopy.

42. The method of claim 39, wherein the label is a colored species, and is visually detectable.

43. The method of claim 39, wherein the label is detectable by virtue of an altered refractive index relative to the unlabeled biomolecule.

44. The method of claim 1, wherein steps (a) through (c) are successively repeated with a plurality of different biomolecules.

45. A method for identifying a compound capable of inhibiting formation of a crystalline structure of a selected biomolecule that is endogenous to the human body, wherein *in vivo* formation of the crystalline structure is an adverse event resulting in at least one medical pathology selected from diseases, disorders, and other undesirable physiological conditions, the method comprising:

(a) providing a combinatorial library of a plurality of different candidate compounds each attached to a different site on a substrate;

(b) contacting the library of candidate compounds with the selected biomolecule under conditions allowing the biomolecule to contact each of the candidate compounds;

(c) identifying candidate compounds for which the biomolecule has affinity by determining which candidate compounds have become physically associated with the biomolecule during step (b);

(d) selecting the candidate compounds identified in step (c) as potential inhibitors of *in vivo* formation of the crystalline structure of the biomolecule;

(e) for at least one candidate compound selected in step (d), contacting the biomolecule with the selected candidate compound under conditions effective to facilitate formation of said crystalline structure in the absence of the selected candidate compound; and

(f) determining whether the crystalline structure is formed in step (e), and if the crystalline structure has not been formed, identifying the selected candidate compound as a compound capable of inhibiting formation of said crystalline structure.

46. The method of claim 45, wherein steps (e) and (f) are successively conducted with each of the candidate compounds selected in (d).

47. The method of claim 45, wherein the plurality of different candidate compounds is selected so that each compound is structurally similar but nonidentical to the biomolecule.

48. The method of claim 47, wherein formation of the crystalline structure comprises formation of fibrils.

49. The method of claim 47, wherein formation of the crystalline structure comprises formation of ocular cataracts.

50. The method of claim 45, wherein the biomolecule is selected from the group consisting of peptidic molecules, sterols, uric acid, uric acid salts, and calcium salts.

51. The method of claim 50, wherein the biomolecule is a peptidic molecule.

52. The method of claim 51, wherein the candidate compounds are independently selected from the group consisting of:

(a) oligopeptide fragments contained within the peptidic molecule; and

(b) analogs of the peptidic molecule wherein the peptidic molecule is modified by (i) substitution of one or more amino acids, (ii) deletion of one or more amino acids, (iii) insertion of one or more amino acids, (iv) an N-terminal modification, (v) a C-terminal modification, or combinations thereof.

53. The method of claim 48, wherein the biomolecule is a peptidic molecule.

54. The method of claim 53, wherein the candidate compounds are independently selected from the group consisting of:

(a) oligopeptide fragments contained within the peptidic molecule; and

(b) analogs of the peptidic molecule wherein the peptidic molecule is modified by (i) substitution of one or more amino acids, (ii) deletion of one or more amino acids, (iii) insertion of one or more amino acids, (iv) an N-terminal modification, (v) a C-terminal modification, or combinations thereof.

55. The method of claim 49, wherein the biomolecule is selected from the group consisting of peptidic molecules, sterols, uric acid, uric acid salts, and calcium salts.

56. The method of claim 55, wherein the biomolecule is a peptidic molecule.

57. The method of claim 56, wherein the candidate compounds are independently selected from the group consisting of:

(a) oligopeptide fragments contained within the peptidic molecule; and

(b) analogs of the peptidic molecule wherein the peptidic molecule or oligopeptide fragments thereof are modified by (i) substitution of one or more amino acids, (ii) deletion of one or more amino acids, (iii) insertion of one or more amino acids, (iv) an N-terminal modification, (v) a C-terminal modification, or combinations thereof.

58. The method of claim 53, wherein the biomolecule is selected from the group consisting of amyloid and amyloid-β.

59. The method of claim 58, wherein the biomolecule is amyloid-β.

60. The method of claim 59, wherein the amyloid-β is selected from the group consisting of amyloid-β (1-40), amyloid-β (1-42), and amyloid-β (1-43).

61. The method of claim 60, wherein the candidate compounds are independently selected from the group consisting of:

(a) oligopeptide fragments contained within amyloid-β; and

(b) amyloid-β analogs wherein amyloid-β or oligopeptide fragments thereof are modified by (i) substitution of one or more amino acids, (ii) deletion of one or more amino acids, (iii) insertion of one or more amino acids, (iv) an N-terminal modification, (v) a C-terminal modification, or combinations thereof.

62. The method of claim 61, wherein the candidate compounds are oligopeptide fragments contained within amyloid-β.

63. The method of claim 53, wherein the biomolecule is prion protein (PrP) or an oligopeptide fragment thereof.

64. The method of claim 63, wherein candidate compounds are independently selected from the group consisting of PrP analogs wherein PrP or oligopeptide fragments thereof are modified by (i) substitution of one or more amino acids, (ii) deletion of one or more amino acids, (iii) insertion of one or more amino acids, (iv) an N-terminal modification, (v) a C-terminal modification, or combinations thereof.

65. The method of claim 63, wherein the biomolecule is PrP.

66. The method of claim 63, wherein the biomolecule is a PrP fragment.

67. The method of claim 66, wherein the PrP fragment is PrP96-111.

68. The method of claim 53, wherein the biomolecule is fibrillar collagen.

69. The method of claim 53, wherein the biomolecule is fibrillin.

70. The method of claim 51, wherein the biomolecule is a lenticular protein.

71. The method of claim 70, wherein the biomolecule is selected from beta, gamma, alpha crystallins, and combinations thereof.

72. The method of claim 71, wherein the biomolecule is selected from beta and gamma crystallins.

73. The method of claim 71, wherein the biomolecule is comprised of a mixture of beta and gamma crystallins.

74. The method of claim 72, wherein the biomolecule is selected from the group consisting of β_H-crystallins, γS-crystallins, γC-crystallins, γD-crystallins, and combinations thereof.

75. The method of claim 51, wherein the biomolecule is cystic fibrosis transmembrane conductance regulator ("CFTR") protein.

76. The method of claim 51, wherein the biomolecule is a Charcot-Leyden protein.

77. The method of claim 51, wherein the biomolecule is selected from the group consisting of cystine, hemoglobin, hematoidin, cryoglobulins, and immunoglobulins.

78. The method of claim 50, wherein the biomolecule is a sterol.

79. The method of claim 78, wherein the biomolecule is cholesterol, cholesterol monohydrate, or a cholesteryl ester.

80. The method of claim 55 wherein the biomolecule is a calcium salt.

81. The method of claim 80, wherein the calcium salt is calcium oxalate, calcium oxalate monohydrate, hydroxyapatite, octacalcium phosphate, tricalcium phosphate, calcium hydrogen phosphate dihydrate, calcium pyrophosphate, or calcium pyrophosphate dihydrate.

82. The method of claim 55, wherein the biomolecule is uric acid or a salt thereof.

83. The method of claim 82, wherein the biomolecule is monosodium urate or monosodium urate monohydrate.

84. The method of claim 45, wherein the biomolecule is labeled with a detectable label, and step (c) is carried out by detecting the presence of the label at any of the different sites on the substrate.

85. The method of claim 84, wherein the label is a fluorescent label, and is detected using fluorescence spectroscopy.

86. The method of claim 84, wherein the label is detectable using ultraviolet spectroscopy.

87. The method of claim 84, wherein the label is a colored species, and is visually detectable.

88. The method of claim 84, wherein the label is detectable by virtue of an altered refractive index relative to the unlabeled biomolecule.

89. The method of claim 45, wherein steps (a) through (c) are successively repeated with a plurality of different biomolecules.

90. A therapeutic agent comprising the selected candidate compound identified in step (f) of claim 45.

91. A method for treating a patient afflicted with a medical pathology associated with *in vivo* formation of a crystalline structure of a biomolecule that is endogenous to the human body, the method comprising:

administering to the patient a therapeutically effective amount of the compound of claim 47.

92. The method of claim 91, wherein formation of the crystalline structure comprises fibril formation.

93. The method of claim 91, wherein the medical pathology is cataract formation, and the biomolecule is a lenticular protein.

94. A method for preventing or treating cataract formation in the eye of a human patient, comprising administering to the patient a therapeutically effective amount of an active agent effective to inhibit crystallization of at least one lenticular protein.

95. The method of claim 94, wherein the active agent is administered in an ophthalmic formulation to the patient's eye.

96. The method of claim 94, wherein the active agent is orally active, and is orally administered in a formulation suitable for oral drug administration.

97. The method of claim 94, wherein the at least one lenticular protein is selected from the group consisting of beta crystallins, gamma crystallins, alpha crystallins, and combinations thereof.

98. The method of claim 97, wherein the at least one lenticular protein is selected from the group consisting of βH-crystallins, γS-crystallins, γC-crystallins, and γD-crystallins, and combinations thereof.

99. A method for simultaneously screening a plurality of candidate compounds for their ability to inhibit formation of a pathogenic mass composed of a crystalline structure of at least one molecular component and associated with a particular medical pathology, the method comprising:

(a) preparing a particulate suspension of the at least one molecular component of the pathogenic mass, by obtaining a pathogenic mass that has been freshly removed from a human patient, lyophilizing and triturating the freshly removed mass, and admixing the lyophilized, triturated mass with an inert organic solvent;

(b) contacting a combinatorial library of candidate compounds with the particulate suspension under conditions effective to facilitate formation of said crystalline structure in the absence of any crystalline structure formation inhibitors; and

(c) identifying candidate compounds for which one or more molecular components of the pathogenic mass have affinity by determining which candidate compounds have become physically associated with a component of the particulate suspension during step (b).

100. The method of claim 99, wherein the pathogenic mass is a cataract.

101. The method of claim 99, wherein the pathogenic mass is atherosclerotic plaque.

102. The method of claim 99, wherein the pathogenic mass is neuritic plaque.

103. The method of claim 99, wherein the pathogenic mass is dendritic plaque.

104. The method of claim 99, wherein the pathogenic mass is a gallstone.

105. The method of claim 99, wherein the pathogenic mass is a kidney stone.

106. A method for identifying a compound capable of inhibiting formation of a pathogenic mass composed of a crystalline structure of at least one molecular component and associated with a particular medical pathology, the method comprising:

(a) carrying out the method of claim 99;

(b) for at least one candidate compound identified in step (c), contacting the particulate suspension with the selected candidate compound under conditions effective to facilitate formation of said crystalline structure in the absence of any crystalline structure formation inhibitors; and

(c) determining whether the crystalline structure is formed in step (e), and if the crystalline structure has been formed, identifying the selected candidate compound as a compound capable of inhibiting formation of the pathogenic mass.

107. The method of claim 106, wherein the pathogenic mass is a cataract.

108. The method of claim 106, wherein the pathogenic mass is atherosclerotic plaque.

109. The method of claim 106, wherein the pathogenic mass is neuritic plaque.

110. The method of claim 106, wherein the pathogenic mass is dendritic plaque.

111. The method of claim 106, wherein the pathogenic mass is a gallstone.

112. The method of claim 106, wherein the pathogenic mass is a kidney stone.

113. A method for identifying a compound capable of inhibiting formation of a crystalline structure of a selected biomolecule that is endogenous to the human body, wherein *in vivo* formation of the crystalline structure is an adverse event resulting in at least one medical pathology selected from diseases, disorders, and other undesirable physiological conditions, the method comprising:

(a) preparing a potential inhibitor by chemically modifying the biomolecule with heat and/or a chemical reactant so as to provide an analog of the biomolecule having at least one difference in molecular structure relative to the unmodified biomolecule;

(b) contacting the biomolecule with the analog under conditions effective to facilitate formation of said crystalline structure in the absence of any crystalline structure formation inhibitors; and

(c) determining whether the crystalline structure is formed in step (b), and if the crystalline structure has not been formed, identifying the analog as a compound capable of inhibiting formation of said crystalline structure.

114. The method of claim 113, wherein steps (a) through (c) are repeated in succession.